CASE REPORT

Aseptic bone necrosis in an amateur scuba diver

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A case is reported that provides further evidence of an old occupational hazard, dysbaric osteonecrosis, presenting in a new population (sports scuba divers) who also appear to be at risk. It highlights the need for an accurate diagnosis of diving related illness.

Amateur scuba diving is a rapidly growing sport. The Professional Association of Diving Instructors (PADI) has reported training over 10 million divers in the last five years.[1] Within the United Kingdom, three organisations, the PADI, the British Sub-Aqua Club, and the Sub-Aqua Association, have over 100 000 active members. Some clubs enrol children as young as 8. Enthusiastic amateur divers are likely to carry out more dives a year than many full time professionals.[2] Changes in technology may affect how divers conduct their dives, thereby altering the risks to which they are exposed. We report here a third case of dysbaric (avascular) bone necrosis (ABN) in an amateur diver with no other known risk factors. ABN is usually associated with prolonged and repeated exposure to pressure and may lead to joint dysfunction and life long disability. It is notable from a previous report of three cases after a submarine escape that one single exposure to pressure may be all that is required to trigger this condition.[3] In that report the disabled submarine was in 37 m of sea water, and the submariners were exposed to increasing pressure for between 2 hours 22 minutes and 3 hours 15 minutes before making their escape. Five of the six escapees survived, but reported suffering from limb bends; 12 years later, three where found to have ABN.

CASE REPORT

A previously fit and healthy 35 year old scuba diver presented to his local accident and emergency department. He had been diving the previous day and was now complaining of his legs ''feeling woolly'', pins and needles in his toes, and poor hand-eye coordination the previous evening. Given the classic symptoms of decompression illness (DCI) and a recent history of diving, a provisional diagnosis of DCI was made. He was placed in 100% oxygen and transferred to our hospital's recompression facility for further care.

On arrival, his symptoms were unchanged, but close questioning revealed that he had been complaining of pain and restricted movement in his left shoulder for several months. Detailed neurological examination was normal. Heel to toe walking and sharpened Romberg time were within normal limits. A diagnosis of acute DCI was made and a decision taken to treat with therapeutic recompression.

In view of the patient's long standing shoulder complaints, a radiograph of the shoulder joint was taken before recompression treatment. The radiograph (fig 1) showed ABN as an advanced necrotic humeral head (A4b MRC classification).[4] Treatment was started using the United States Navy recompression treatment table 6. The recompression treatment was uneventful, and the patient made a full recovery from acute DCI.

During follow up, the patient was offered a radiographic long bone survey, and magnetic resonance imaging (MRI) of any lesions found. Radiographs revealed subtle ''snow capping'' of the right humeral head with increased subchondral density typical of early ABN (fig 2). MRI detailed the extent of the damage underlying the bony changes seen on plain radiographs (figs 3 and 4).

DISCUSSION

Radiographic examination is not routine in assessing diving illness. However, when making a diagnosis one needs to exclude other causes, such as trauma, so as not to treat symptoms that do not origin from acute diving illness, thereby avoiding the expense and inconvenience of protracted recompression treatment. We identified evidence of ABN that accounted for the shoulder pain. The extent of the damage was striking in the light of the patient's continued use of the limb, his occupation as a fireman, and pursuit of arduous physical sport.

The patient has no history or known medical causation of ABN other than hyperbaric exposure—for example, diabetes, steroid use, hyperlipidaemia, or trauma. He is a non-smoker, and consumes less than 15 units of alcohol a week. As an experienced diver, he was conducting sports dives to the limits of technology—that is, relatively deep dives using multiple gas mixtures rather than depth limiting air as the breathing medium. Although such deep diving practice is currently restricted to the more adventurous, advancements in technology and equipment are making such dives more commonplace.

ABN is a well documented hazard of pressurised tunnel work and to a lesser extent professional diving. It was first reported in 1911 by Bassoe.[5] When associated with pressure

Figure 1 Plain radiograph of left shoulder, showing established avascular bone necrosis of the proximal humerus with collapse of the articular surface.

Abbreviations: ABN, avascular bone necrosis; DCI, decompression illness; DON, dysbaric osteonecrosis; MRI, magnetic resonance imaging

Figure 2 Plain radiograph of right shoulder, showing subtle ''snow capping'' with increased subchondral density typical of early avascular bone necrosis.

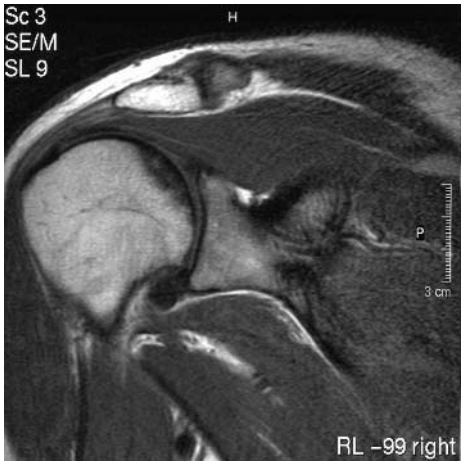

Figure 3 Magnetic resonance imaging scan of the right shoulder. T1 weighted image indicating loss of subchondral signal showing a geographic pattern corresponding to the ''snow capping'' seen on the plain radiograph.

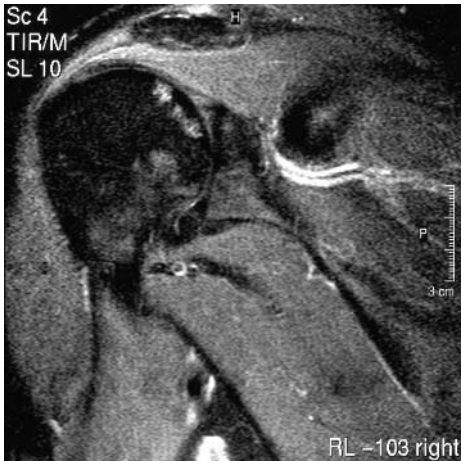

Figure 4 Magnetic resonance imaging scan of right shoulder. T2 weighted image, with fat suppression revealing early avascular bone necrosis changes. White area indicates subchondral oedema corresponding to the geographic pattern seen on plain radiographs.

Take home message

- Diving related illness can present with chronic lesions as well as the paradigm of symptoms commonly referred to as the ''bends''. Clinicians who examine divers for whatever reason should be aware of this old occupational hazard presenting in a new population who also appear to be at risk.
- There have been no studies to establish the incidence and prevalence of this potentially disabling condition in amateur scuba divers. Epidemiological surveys are justified.

exposure, it is often referred to as ''dysbaric osteonecrosis'' (DON). Ohta and Matsunga[6] and Wade *et al*[7] reported an incidence of DON greater than 50% in discrete populations of professional shell fishermen in Japan and Hawaii who conduct deep scuba dives. When it affects the articular surfaces of the joints, it can lead to disablement of the affected limb. Its initial presentation can be painless necrosis of the joint detectable only by MRI, radiography, or similar diagnostic tool. It can occur in divers and tunnel workers who have not reported experiencing acute DCI. There are only three previous case reports in the world literature of DON in amateur scuba divers.[8–10] In one of the reports, the diver had a history of insulin dependent diabetes and joint trauma, thus detracting from hyperbaric exposure as the sole causation. DON is believed to result from inadequate decompression.[11] There is convincing evidence that pathologically DON is the result of nitrogen and lipid emboli initiating a coagulation cascade in both tissue and the microcirculation of bone.[12] There are reports of other ''diving'' related causes, including theories about the compression/decompression cycles. There is, however, little direct supporting evidence.[13] DON can occur months or years after exposure to increased atmospheric pressure. It appears that only one pressure exposure is needed to trigger the condition.

Wilmshurst[14] reported that advancement of technology does not always advance the safety of sports divers. With increasingly modern equipment and changing life styles, amateur divers go deeper for longer, more often. Perhaps more worrying, amateur divers have a no (or low) risk perception of DON, a potentially disabling condition.

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